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PROLAPSE OF INVAGINATED COLON THROUGH THE ANUS IN GOLDEN HAMSTERS (*MESOCRICETUS AURATUS*)[1,2]

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SUMMARY • *Six cases of invagination of the colon with prolapse through the rectum were reported in the Golden Syrian hamster. The anatomy of the colon and its enveloping mesentery in the hamster are thought to be predisposing factors in the process of invagination. The mesenteric fold incorporates the spleen in this animal and acts as a ligament stopping further invagination. The gross pathology associated with the process of invagination was described. Observations with respect to the pathogenesis were included, emphasizing the point that peristalsis and not the initiating intestinal irritant is the major causal factor.*

KEY WORDS • *Hamster—Colon—Prolapse—Invagination*

The most common locations for intestinal invaginations are in the ileum, jejunum, and cecum (1, 2). This process occurs in all species of animals, but is most common in dogs, young horses, adult cattle, and lambs (2).

The cause of intestinal invaginations has not been clearly defined (2). However, enteritis, increased peristalsis, dysperistalsis, pedunculated tumors projecting into the lumen, foreign bodies, and parasites have all been found associated with invaginations (1–6). Intestinal irritations secondary to infectious diseases (*ie*, canine distemper) have also been incriminated, as has the introduction of cold foods and/or fluids (1).

Invagination of the colon with prolapse through the rectum (not to be confused with simple prolapse of the rectum and anus) is a much less common finding in all species (1–6).

This is a report of invagination of the colon which was observed in 6 hamsters from 6 groups of 20 each during an 11-mo period.

FINDINGS

One hundred-twenty young male golden Syrian hamsters Lak:LVC(SYR) 90–110 g, were housed individually in galvanized cages. Water and laboratory diet[4] were supplied *ad libitum*. The hamsters discussed in this paper were from groups receiving intragastric inoculations of live *Salmonella* organisms (10^8–10^9 organisms/ml/50-g body weight) as part of studies of the mechanisms of food poisonings. Problems evidenced by these hamsters occurred at intervals over 11 mo.

The hamsters were held for observation for 1 wk and then placed on test for 2 wk. There was no evidence of naturally-ocurring diarrhea in any of the animals prior to the test. *Salmonella* organisms were recovered from all test animals. One of the 6 hamsters was found dead in its cage in the morning. The only sign of disease grossly visible was protrusion of 1–1.5 cm of colon from the anus (Fig 1). Each of the other 5 hamsters was found with a colonic protrusion from the anus prior to death. Three of these had blood and debris caked around the nose and mouth, and

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[2] In conducting the research described in this report, the investigator adhered to the "Guide for Laboratory Animal Facilities and Care" as promulgated by the Committee on the Guide for Laboratory Animal Resources, National Academy of Sciences—National Research Council, and by the National Society for Medical Research.
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[4] Purina Laboratory Chow®. St Louis, MO.

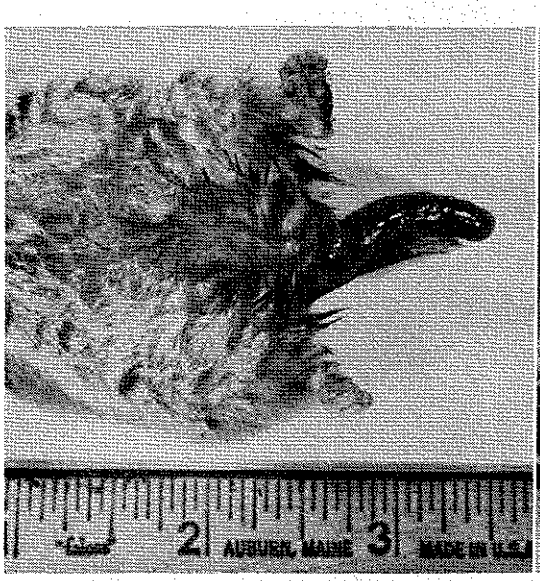

Fig 1. *Anesthetized hamster with protruding colon.*

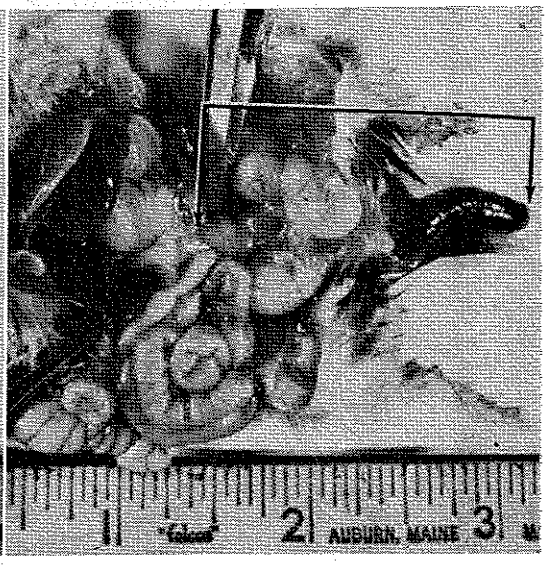

Fig 2. *Same hamster showing extent of invagination.*

the protruding segments of colon had evidence of self-inflicted trauma. They also had watery diarrhea. There was no evidence of self-trauma in the other 2 hamsters. Three hamsters were killed within 1 hr of discovery and necropsied. The remaining 2 hamsters developed classical signs of intestinal obstruction, *ie*, distended abdomen, dehydration, anorexia, and pain on palpation before they were killed and necropsied. These signs appeared in 1 hamster within 30 hr and in the other within 48 hr (time from discovery to killing). By this time, the protruding intestine had become edematous and necrotic (Fig 2). Other hamsters in each test group displayed varying degrees of clinical signs associated with salmonellosis but were not afflicted with the colonic invagination.

The necropsy findings were similar in the 6 animals. The colon was distended with ingesta and gas proximal to the point of invagination. Generalized petechiae and echychmoses were evident in the colonic wall. The distal segment (descending colon) and the invaginated segment were edematous and necrotic. Fibrinous adhesions were evident when traction was applied to remove the invaginated segment. In all 6 hamsters the invagination process had proceeded to a point corresponding to the junction of the transverse and descending colon. At this point in the hamster, there is a fold of mesentery that incorportes the spleen and effectively acts as a ligament binding the spleen and this junction of the colon together. The lengths of the descending colon varied from 4.8–6.2 cm. Thus, the invaginated portion of the colon traveled distances of approximately 6–8 cm before the process was terminated (*ie*, either by death, killing, or the holding capacity of the spleno-colic-mesenteric ligament).

Discussion

Invagination of the colon with protrusion through the anus has rarely been described. The process is probably uncommon in most animals. However, the hamster is unique in anatomical configuration with respect to the colon. The transverse colon of the hamster is very long (up to 8 cm), while the ascending colon is more than twice as long (up to 21 cm). Further, instead of being a straight tube,

the ascending colon winds tortuously towards the junction with the transverse colon with several foldings on itself in its mesenteric sheath. The fold of mesentery incorporating the spleen and colon at the junction of the transverse and descending colon is capable of acting as ligamentous attachment to the colon at this point, preventing further invagination. To invaginate further would have the effect of incorporating the spleen into the invagination.

As noted earlier, the list of suggested causes associated with invagination of the intestine includes infections and other irritants. In this case, the author believes that the infection was the irritant. However, regardless of the initiating irritant, peristalsis is the major operator. At some point in the intestine, spasm of the circular muscle occurs, produced by a violent peristaltic motion (1). A following peristaltic wave in the longitudinal muscle carries the spastic portion into the lumen of the distally-relaxed intestine (1). It is this writer's opinion that the hamster is predisposed to this type of intestinal invagination process in an area 6–8 cm proximal to the junction of the transverse and descending colon, and that the spleno-colic-mesenteric fold acts as a ligament preventing further invagination.

Several points are worthy of mention here: 1) the hamster appears to be more susceptible to invagination processes in the colon than in the small intestine, as opposed to most other species in which the reverse is true; 2) investigators wishing to maintain animals for long-term experiments should be cautioned against assuming that they are dealing with a simple anal-rectal prolapse that is correctible via simple replacement and perianal purse-string suture. Colonic invagination must be considered in the differential diagnosis; and 3) future investigators might consider the hamster a useful biologic model in working with problems associated with intestinal invagination processes.

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